Amendments to the Specification:

Please amend the specification as set forth below. Deletions are indicated by a strike through and insertions are indicated by double underlining.

Please delete the paragraph beginning at page 7, line 28, and insert the following replacement paragraph:

Figure 1 illustrates the co-stimulatory effects of CTL activity elicited by coordinate administration of a ~~B7-1-encoding~~ B7.1-encoding vector with an HPV E7 peptide antigen, as compared with the immunogenic response elicited by the E7 peptide antigen alone. C57B1/6 mice were vaccinated with emulsion containing E7 peptide with or without co-administration of a B7.1-encoding DNA vector. B7.1 DNA was administered, either in the same site as the E7 peptide or on the other side of the base of the tail, as indicated. The figure shows that coordinate administration of the B7.1-encoding DNA vector significantly enhances the E7-specific ~~CTl~~ CTL response when the vector and peptide are delivered to a common target site.

Please delete the paragraph beginning at page 13, line 3, and insert the following replacement paragraph:

Ras is among the best characterized oncogenes in human cancers ~~is *ras*~~ (Boa et al., Nature 327:293-297, 1987; Satoh et al., Semin. Cancer Biol. 3:169-177, 1992; and Grand et al., Biochemistry 279:609-631, 1991, each incorporated herein by reference). A single point mutation in codon 12 of the *ras* gene accounts for more than 90% of all *ras* mutations (Hruban et al., Am. J. Pathol. 143:545-554, 1993; Li et al., Am. J. Pathol. 144:303-309, 1994; and Breivik et al., Br. J. Cancer 69:367-371, 1994, each incorporated herein by reference), and is present in more than 20% of all solid tumors (i.e., more than 800,000 cancer patients in the United States). In addition, the Ala^{59}, Gly^{60} and Gln^{61} residues of the *ras* proto-oncogene are frequently mutated in human tumors (Chung et al., Science 259:806-809, 1992, incorporated herein by reference).

Please delete the paragraph beginning at page 13, line 13, and insert the following replacement paragraph:

Therefore, mutant *ras* peptides serve as particularly useful vaccine agents to elicit anti-cancer immune responses according to the methods of the invention. In this context, *Ras* p21 is an intracellular protein subject to ~~antigenprocessing~~ antigen processing and presentation by MHC molecules. Specific CD4+ and CD8+ T lymphocytes that can recognize a single *ras* mutation have been described. Murine experiments have shown that T lymphocytes specifically immunoreactive against mutated *ras* peptides have the ability to lyse target cells that endogenously express the same point mutated *ras* gene. These lytic T cells display cytotoxic activity of both CD4+ (Th1 subtype) and CD8+ subsets (Abrams et al., Eur. J. Immunol. 25:2588-2597, 1995; Peace et al., J. Immunol. 14:110-114, 1993; Peace et al., J. Exp. Med. 179:473-479, 1994; and Skipper et al., J. Exp. Med. 177:1493-1498, 1993, each incorporated herein by reference). Furthermore, induction of anti-*Ras* CTLs by vaccinating mice with recombinant mutant *ras* proteins has led to the rejection of syngeneic tumor cells bearing the corresponding mutation (Fenton et al., J. Natl. Cancer Inst. 85:1294-1302, 1993, incorporated herein by reference).

Please delete the paragraph beginning at page 21, line 9, and insert the following replacement paragraph:

In relation to more detailed aspects of the invention, studies have been conducted testing 9, 10 and 11 residue peptides derived from HIV p18, overlapping or contained within the p18-I-10 peptide, including specifically both possible 9 residue peptides contained within p18-I-10, and all of these have been found to be less active than p18-I-10. This finding concerning the importance of length in the activity of peptides presented by ~~NBC~~ MHC class I molecules and the identification of a truncation of p18, p18-I-10 (residues 318-327), with 10 to 10^2-fold greater potency of T-cell stimulation provides general guidance for selecting other peptide or protein antigens for use within the invention.

Please delete the paragraph beginning at page 38, line 13, and insert the following replacement paragraph:

~~additional~~ Additional segments that provide for its transcription. As noted above, such additional segments include promoter and terminator sequences. DNA vectors for use within the invention also may include one or more origins of replication, one or more selectable markers, an enhancer, a polyadenylation signal, etc. Expression vectors generally are derived from plasmid or viral DNA, and can contain elements of both. The term "operably linked" indicates that the segments are arranged so that they function in concert for their intended purposes, for example, transcription initiates in the promoter and proceeds through the coding segment to the terminator (see, e.g., Sambrook et al., Molecular Cloning: A Laboratory Manual, Cold Spring Harbor Laboratories, Cold Spring Harbor, N.Y. 1989, incorporated herein by reference).